Necrose aguda do esófago (Esófago negro)

Acute esophageal necrosis (Black Esophagus)

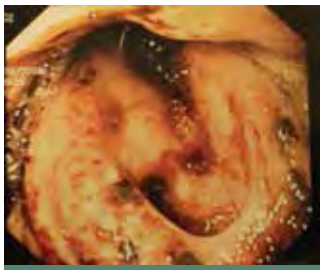
Alda Garcia, Tereza Patrícia, M.C. Perloiro

51 years old woman, previously autonomous with a health background of type 2 Diabetes Mellitus, evolving for over 10 years and a poor previous control, without alcoholic habits or reference to the intake of caustic substances, medicated only with insulin in the outpatient clinic. She arrived at the Emergency Service with hematemesis, abdominal pain and vomiting for 5 hours.

She was aware, hemodinamically stable with ketonic breath, without other changes relevant for the objective exam. The test results revealed Hb 10.5g/dL, arterial gasometry with metabolic acidemia (pH 7.01) and ketonuria. Upper gastrointestinal endoscopy (UGIE) has shown an esophagus with continuity patches of pearly color in the mucosae, with a circumferential distribution up to the level of esophagus--stomach transition and areas of longitudinal necrosis in the most distal portion (*Fig. 1. and 2*), no biopsies were carried out. The diagnosis hypothesis of acute esophageal necrosis (AEN) was raised.

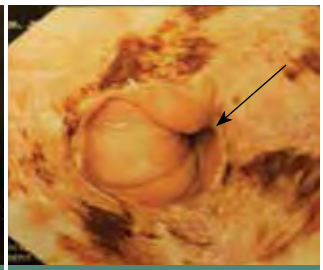
During hospitalization and after compensating the diabetes, the patient evolved favorably without hematemesis reoccurrence and tolerating oral feeding. Control UGIE on the 14th day did not reveal any lesion.

Acute esophageal necrosis (AEN) was described for the first time in the endoscopy era by Goldenberg as previously the diagnosis was only made in the postmortem histologic exam.[1] The etiology is unknown but pathophysiological mechanisms have been suggested as ischemia, gastro-esophageal reflux, alcohol, iatrogenic events and viral or fungal infections. There are AEN described cases in patients with diabetic ketoacidosis, metabolic acidemia, hypovolemic shock or in the post-surgical period in major surgeries.[1,2] The endoscopy findings are characterized by a black esophageal mucosae, friable and hemorrhagic with a preferential involvement of the distal esophagus, with the mucosa change ending abruptly in the esophagus-gastric transition. The histology is important for the differential diagnosis with melanosis, pseudomelanosis, melanoma or acanthosis nigricans, progressing with a darkening mucosa but without necrosis. AEN most important complication are esophageal perforation with a reported incidence below 7% and esophageal stenosis, described in over 10% of patients.[3;4] AEN treatment consists in: adequate hydration; feeding pause lasting at least 24 hours; proton pump inhibitors, sucralfate and in the treatment of comorbidities. The prognosis depends on the underlying clinical condition, age and comorbidities. ■



Esophagus with areas of necrosis.

FIG. 1



Esophageal-gastric transition.

FIG. 2

Medicine II Service of Hospital Fernando Fonseca

Received for publication on the 29th October 2010
Accepted for publication on the 16th June 2011

References

1. Goldenberg SP, Waint, Marignani P. Acute necrotizing esophagitis, Gastroenterology 1990:98:493-496.

2. Augusto F. Fernandes et al. Acute necrotizing esophagitis: a large retrospective case series, Endoscopy 2004; 36: 411-415.

3. Grigoriy E Gurvits et al. Black esophagus: Acute esophageal necrosis syndrome, World S. Gastroenteral 2010; 16(26): 3219-3225.

4. Grigoriy E Gurvits et al. Acute esophageal necrosis: a rare syndrome. S.Gastroenteral 2007; 42:29-38.